

The Lack of Environmental Justice in Central and Eastern Europe

The conclusion of the recent *EHP* monograph on environmental justice (Shepard et al. 2002) is that environmental exposures and environmental health issues impact disproportionately on vulnerable populations. In Central and Eastern Europe, we enviously read about the studies (primarily American) carried out in this field. As a consequence of the lack of moral, political and financial support, this type of research is currently not possible in this area of Europe.

In fact, Central and Eastern Europe would be an excellent model area for this type of study. The total area of six to eight countries in Central and Eastern Europe is equivalent to that of one of the smaller states in the United States. Because the countries are small, environmental pollution easily crosses the borders, which are most frequently political and are rarely geographical or hydrological (i.e., natural). The pollution often affects the health of populations of adjacent countries. Sometimes environmentally irresponsible behavior is a consequence of the past. Unfortunately, articles that focus on the historical and political background of current environmental situations in Central and Eastern Europe are not frequently found in the literature. [At this point the reader may ask why we do not write one. We did write an article about the Carpathian Basin (Varga et al. Unpublished data), but *EHP* would not publish it because of the political nature of the manuscript.] However, an article that does not investigate the true causes of the problems is meaningless. In other words, it is not the articles, but the problems themselves, that are saturated with politics. In order to solve the problems, it is necessary to face them head on.

Faber and Krieg (2002) suggested that, in Massachusetts, the "ecologically hazardous sites and facilities are disproportionately located and concentrated in communities of color and working-class communities." This is also true for other locations in the United States. There are many similar examples in Central and Eastern Europe, such as a recent scandal in Hungary: After the country's largest sports hall was destroyed by fire, the debris containing crocidolite asbestos was dumped illegally in an empty site close to a working-class housing district in Budapest.

In some countries in Central and Eastern Europe, environmentally hazardous sites and activities are also disproportionally located, with high concentrations in the areas and communities of ethnic or national minorities. In the majority of countries in Central and Eastern Europe, the classical term of ethnic minority predominantly means the

Romany (gypsy) population. For example, in the case of a Hungarian Romany community in Heves, where 1,500 persons were exposed to high doses of lead as a consequence of the illegal disassembly of car batteries collected from nearby dumps and gas stations, one 15-month-old girl died and 65 children and 14 adults were hospitalized (Varró et al. 2001).

National minorities are often subjects of environmental injustice. These minorities are primarily products of political and governmental changes in the twentieth century, when millions of people suddenly found themselves living in a different country while still in their own homes, depending on the interests of great powers. That is, because the region where they lived was absorbed by another country, the residents frequently became second class citizens of their new state. This has been a common occurrence (both as enclaves and as compact zones along the borders) from the Baltic Sea to the Balkans (Brown 1999, Varga and Ember 2000).

Manipulated industrialization was a characteristic feature of the fallen national communist regimes. These environmental "hot spots" exist today, exposing residents of several countries to long-term or permanent hazards. Neighboring countries can be exposed to potential environmental risks; for example, there are international debates on nuclear power plants (e.g., Czech Republic vs. Austria, Slovakia vs. Austria and Hungary); atmospheric pollution (e.g., Poland vs. Germany), and river pollution (upstream vs. downstream countries). Research in environmental justice is urgently needed to aid in the resolution of environmental issues.

Both scientifically and politically unique issues have also arisen in Central and Eastern Europe. The recent environmental catastrophe in the Carpathian Basin was caused by an Australian–Romanian joint venture in Baia Mare/Nagybánya, Romania, inhabited primarily by ethnic Hungarians. The company used (and currently uses) hazardous technology for gold production, which is not allowed in many other countries (e.g., Australia). In January 2000, the mine discharged almost 100,000 m³ of concentrated cyanide solution and polluted the Hungarian section of the Tisza River through the Szamos catchment area. The total quantity of cyanide (in the pollution wave) was approximately 105–110 tons; 70–100 tons of copper was also detected. This pollution almost completely killed the plankton in the Tisza and Szamos Rivers. The most spectacular consequence of this cyanide pollution, however, was an enormous fish kill, estimated at 1,241 tons. Fortunately, drinking-water production for the involved cities was stopped in time to prevent massive human

exposure (Standovár and Primack 2001). In this case, Romanian producers with Australian capital caused both ecologic and economic damage as well as a health hazard for the Hungarian population. [Indeed, new mines with the same technology are planned (e.g., Verespatak project with Canadian capital). It is a new type of eco-colonialism.] To date, both Hungary and the company are still debating on which country's laws should be used in the compensation trial. Meanwhile, the joint venture was cleared in a criminal suit in the Romanian court, but the European Union Inspection Committee declared that the company is responsible for the ecologic catastrophe (European Commission 2001).

Another example is the Danube Dam Project. The goal of the Danube Dam Project, began in the communist era, was to effectively use the Danube River, which in this area forms the border between Slovakia and Hungary. Hungary withdrew from the project following the change of the government to a democracy. The Slovak Academy of Sciences called attention to fact that the project could cause contamination of drinking water in Csallóköz (Slovakian territory north of the Danube with a Hungarian population). The Danube was redirected into an artificial canal in Slovakia to a new hydroelectric power plant, bypassing the Old Danube (the frontier river). This operation caused an extraordinary decrease in surface water, leading to a decrease in the water table in the Hungarian Szigetköz and thus causing branches, channels, and backwaters of the river to dry up, threatening the unique biotopes. Transportation on the river was also redirected to Slovak territory, leading to even more legal disputes. Although the International Court (1997) decided that water distribution should be the subject of bilateral negotiations, to date, the problems still have not been resolved.

This situation with the Danube Dam Project is also special in that ethnic Hungarians live on both the Slovakian and Hungarian sides of the river. This population bears the environmental risks and disadvantages, while others receive the benefits. Severe ecologic issues have already arisen in the Hungarian Szigetköz with drying up of river tributaries, but most of the problems in Hungarian communities in the Slovakian territory (e.g., isolation by the artificial canal) are sociologic in nature. Similar situations may have also occurred in other countries.

Who benefits, and who bears the costs? In Central and Eastern Europe, international dimensions are especially complicated by the political history of the region. Scientific research is needed to address the environmental injustice of international

ecological catastrophes, especially those in Central and Eastern Europe.

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Proposed PBPK Model to Predict Infant Exposure to Toxic Chemicals in Breast Milk

In the mini-monograph, "Pharmacokinetics of Toxic Chemicals in Breast Milk: Use of PBPK Models to Predict Infant Exposure," published in the June issue of *Environmental Health Perspectives*, Clewell and Gearhart (2002) described a model schematic developed in rats for the lactational transfer of perchlorate and iodide from the mother to the neonate. They used this physiologically based pharmacokinetic (PBPK) model to predict the distribution of perchlorate and iodide in lactating mothers and in infants in humans.

It is thought that because of its similarity in ionic size to iodide, the perchlorate ion is a competitive inhibitor of iodide at the sodium iodide symporter (NIS), the plasma membrane protein that catalyzes the accumulation of iodide into thyroid cells (Wolff 1998). The perchlorate ion blocks the binding of iodide to the NIS and is not internalized by the NIS into the thyroid cell (De La Vieja et al. 2000). Perchlorate can accumulate in the thyroid but does not seem to accumulate in

the thyrocytes (Eskandari et al. 1997). Perchlorate is excreted intact in the urine and has a half-life in humans of about 6–8 hr. Approximately 95% is recovered in the urine over 72 hr (Eichler 1929). Perchlorate is excreted quickly and is not stored, but enough iodine is stored in humans for about 6 weeks.

The PBPK model described by Clewell and Gearhart (2002) is a "schematic of the lactation model for the rat and human." The rat model was included in the March 2002 external review draft in preparation for the U.S. Environmental Protection Agency Toxicological Review and Draft Risk Characterization meeting held 5–6 March 2002 in Sacramento, California. At the meeting Nancy Carrasco, whose team sequenced and cloned the NIS (Dai et al. 1996), repeatedly claimed that perchlorate is not internalized by the thyroid cells. Additionally, NIS sites have not been found in human skin (Ajjan et al. 1998; Vayre et al. 1999).

Perchlorate has been in medical use since the 1950s, primarily for the treatment of hyperthyroidism; it is still in clinical use today mainly for diagnostic purposes. The toxicity of perchlorate in humans is known, as is its dose response in humans who have been exposed therapeutically, occupationally, in clinical studies, or environmentally via drinking water (Soldin et al. 2001). Iodide inhibition will not cause adverse effects on humans if thyroid hormone levels remain normal. Assuming a daily intake of 2 L water/day, the highest known level of perchlorate in drinking water (24 µg/L) would yield a daily exposure of less than 50 µg/day. This seems a large range of safety, being 10-fold lower than the 500 µg/day level that Greer et al. (2002) considered to be the no-effect level in humans for the inhibition by perchlorate of iodine uptake by the thyroid, the mode-of-action held to be the initial and essential pharmacologic effect of perchlorate. It is also a thousand-fold lower than the 50 mg/day level at which an effect on thyroid hormone levels in humans may be expected. The absence of an observed effect on neonatal thyroid, thyroidal diseases, or thyroidal cancer in areas with higher detected perchlorate levels is epidemiologically consistent with the human toxicologic and pharmacologic observations.

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Proposed PBPK Model to Predict Infant Exposure: Clewell and Gearhart's Response

In her letter, Soldin makes a number of points regarding the potential human toxicity of perchlorate (ClO₄⁻), one of the chemicals mentioned in our paper (Clewell and Gearhart 2002). The purpose of our article (Clewell and Gearhart 2002) was to describe the potential use of physiologically based pharmacokinetic (PBPK) modeling to address the challenges encountered in calculating chemical transfer in breast milk. We presented ClO₄⁻ as one of several examples of how PBPK modeling can be used to predict neonatal dose where little or no data are available in humans. The model includes descriptions for ClO₄⁻ and iodide in the lactating rat and neonate and the extrapolation of radioiodide to human lactation. We suggested that the successful extrapolation of the rat radioiodide model to predict human milk increases confidence in using a similar PBPK-based approach to estimate infant ClO₄⁻ dose (Clewell and Gearhart 2002).

With respect to the PBPK model for ClO₄⁻, Soldin expresses concern regarding the model structure, stating that ClO₄⁻ is not transported into the thyrocyte. This contention is based solely on *in vitro* electrogenicity studies in frog oocytes (Eskandari et al. 1997) and Chinese hamster ovary cells (Yoshida et al. 2002), the results of which are

susceptible to two explanations: *a*) ClO_4^- blocks I^- transport but is not transferred into the thyroid, and *b*) ClO_4^- competes with I^- and is transported by sodium iodide symporter (NIS) at a 1:1 ratio with Na^+ (Dohan et al. 2000; Nilsson 1999; Riedel et al. 2001a, 2001b). These three studies did not measure thyroid ClO_4^- levels, nor did they offer concrete evidence disproving the translocation of ClO_4^- into the follicle.

Perchlorate transfer into the thyrocyte is evidenced by the effect of ClO_4^- on internalized thyroid iodide (Hildebrandt and Halmi 1981). In the presence of propylthiouracil (which blocks iodide organification) and thyroid stimulating hormone (which increases inorganic iodide uptake), ClO_4^- exposure caused a significant discharge of the internal iodide. This suggests that ClO_4^- enters the thyroid cell and displaces iodide from binding sites in the thyrocyte.

The model description is also supported by measured thyroid:serum ClO_4^- ratios > 1 and up to 30 (Chow and Woodbury 1970; Yu et al. 2002a). These high anion concentrations cannot be explained without active sequestration, and the possibility of analytical interference by metabolites is ruled out by a double-labeled $^{36}\text{Cl}^{18}\text{O}_4^-$ study that showed no appreciable metabolism in the rat (Anbar et al. 1959). Furthermore, time-course data show two distinct phases in ClO_4^- uptake in the thyroid (half-life = 2 and 33 min), which Chow and Woodbury (1970) suggested represent the rapid transport into the interstitial and cellular compartments and the slow equilibration in the luminal fluid, respectively. This same behavior was also observed in the clearance curve from an intravenous dose (Yu et al. 2002a). Thus, when taken together, the weight of evidence suggests that ClO_4^- is a truly competitive inhibitor (Chow and Woodbury 1970; Hildebrandt and Halmi 1981; Wolff 1998; Yu et al. 2002a). Our mode of action hypothesis, which served as the foundation for the kinetic models, was based upon the entire body of available information.

A second concern mentioned by Soldin was the inclusion of active uptake of ClO_4^- in the skin. In the model, the skin compartment actively sequesters ClO_4^- and iodide from the blood but is not a route of entry. This description of ion transport in the skin is supported by the presence of NIS (Kotani et al. 1998) and the accumulation of iodide and ClO_4^- in rat skin (Brown-Grant 1959; Yu et al. 2002a), as well as ClO_4^- -induced inhibition of iodide uptake in the skin of fetal and neonatal rats (Yu et al. 2002b; Zeghal et al. 1995). Because the skin is not the target tissue, it is not critical to prove the mode of ion transport. However, it is important to accurately describe skin con-

centrations, since blood levels and, therefore, milk transfer and thyroid kinetics, could be affected by the transfer into such a large organ.

Finally, Soldin suggests that the dose-response relationship for ClO_4^- toxicity is known from a variety of human exposure scenarios. We contend, however, that quantitative information on dose response in humans is incomplete at best and essentially nonexistent in the newborn. Although thyroid hormones have been measured in ClO_4^- -exposed infants and children in epidemiologic studies (Crump et al. 2000; Lamm and Doemland 1999; Li et al. 2000), neurologic end points have not been correlated to a neonatal ClO_4^- dose. Because ClO_4^- inhibits iodide uptake in the thyroid and in milk (Brown-Grant 1957; Potter et al. 1959; Howard et al. 1996) and is present in the milk of lactating rats (Clewell and Gearhart 2002), it would seem prudent to quantitatively determine the extent of neonatal exposure to ClO_4^- and the resulting inhibition of neonatal thyroid iodide. The PBPK models presented in our paper (Clewell and Gearhart 2002) enable us to develop a reasonable estimate of dose in the population of interest, the human neonate, thereby providing a vital piece of information needed for a more informed human health risk assessment.

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Personal and Political Aspects of MCS

Thank you for the August 2002 *Environmental Health Perspectives Supplement* on "Air Toxics and Asthma" and "Environmental Factors in Medically Unexplained Diseases" [*Environ Health Perspect* 110(suppl 4)]. I am pleased to see the recommendations for research on illnesses such as chronic fatigue syndrome, fibromyalgia, sick-building syndrome, and multiple chemical sensitivities (MCS).

For several years I have had reactions to many scented products, auto exhaust, some plastics and inks, and numerous household chemicals. I have had allergies most of my life, which are well managed by medication. Chemical sensitivities have been more of a hardship than allergies and, to my knowledge, avoidance is the only effective therapy.

Years ago, I had a short-term illness after using furniture stripper, and years later, I had a long-term illness following repeated use of a 12.8% lindane solution. As my sensitivities increased, repeated chemical exposures in daily life brought me chronic and debilitating fatigue, intermittent severe stabbing pain,

earaches, tooth and jaw pain, visual problems, loss of bladder control, loss of coordination, nose bleeds, respiratory problems, disorientation, and vasculitis in my hands and feet.

Since then, I have removed many household cleaning products and personal care products from my home. It is almost impossible to keep heavily scented products from entering the home on packaging, plastics, store items, and clothes worn by visitors. Avoiding chemicals that commonly cause symptoms of illness in sensitive persons is

difficult and violates social norms. Avoidance of irritants has significantly improved my general health and energy level, and accommodation at work has allowed me to avoid newly remodeled areas and scented personal care products of co-workers.

Chemical and pharmaceutical companies have an enormous stake in the medical coding of MCS as a psychologic rather than a physiologic illness. MCS should not be reduced to anticipation of litigation and fiscal liability.

Mental illness is stigmatized in our society and is discriminated against in long-term disability insurance plans as well as in some health insurance plans. It would be unfair to define MCS as a mental illness if the disease is actually caused by chemical injury.

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NIEHS scientists and grantees are performing basic studies of our susceptibility to environment-related disease: demonstrating that a carcinogen in cigarette smoke (benzo(a)pyrene) alters part of a gene to cause lung cancer . . . showing the effects of fetal exposure to PCBs . . . developing a strain of mouse that lacks functional estrogen receptors and that helps evaluate how some pesticides and other estrogen-like compounds might affect development and reproduction . . . discovering the genes for breast, ovarian, and prostate cancers . . . identifying women's optimal days of fertility . . . seeking to reverse the damage from lead exposure . . . finding alternatives to traditional animal tests . . . pinpointing the functions of specific genes by eliminating them from specially bred mouse lines . . . discovering a way, using ordinary yeast cells, to isolate and clone genes and other fragments of genetic material more quickly . . . showing the effects of urban air on lung function . . .

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